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Blood Glucose Control for Individuals with Type-2 Diabetes: Acute Effects of Resistance Exercise of Lower Cardiovascular-Metabolic Stress

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Abstract

Moreira, SR, Simões, GC, Moraes, JFVN, Motta, DF, Campbell, CSG, and Simões, HG. Blood glucose control for individuals with type-2 diabetes: Acute effects of resistance exercise of lower cardiovascular-metabolic stress. *J Strength Cond Res* XX(X): 000–000, 2012–This study compared the effects of resistance exercise (RE) intensities on blood glucose (GLUC) of individuals without (ND) and with type-2 diabetes (T2D). Nine individuals with T2D and 10 ND performed: (a) RE circuit at 23% of 1 repetition maximum (1RM) (RE_L); (b) RE circuit at 43% 1RM (RE_M); and (c) control (CON) session. Blood lactate (LAC) and GLUC were measured before, during, and postinterventions. Double product (DP) and rate of perceived exertion (RPE) were recorded. The area under the curve (AUC) revealed the effects of RE circuits in reducing GLUC in individuals with T2D (RE_L: 12,556 ± 3,269 vs. RE_M: 13,433 ± 3,054 vs. CON: 14,576 ± 3,922 $mg \cdot dl^{-1}.145$ minutes; $p < 0.05$) with a lower AUC of GLUC in RE_L in comparison to RE_M. Similarly, for ND the RE_L reduced the AUC of GLUC when compared with RE_M and CON (RE_L: 10,943 ± 956 vs. RE_M: 12,156 ± 1,062 vs. CON: 11,498 ± 882 $mg \cdot dl^{-1}.145$ minutes; $p < 0.05$). The AUC of GLUC was higher for T2D compared with ND on CON condition ($p = 0.02$). However, after RE circuits the difference between groups for AUC of GLUC was abolished. The RE_M for T2D was more stressful when compared with RE_L for LAC (CON: 1.3 ± 0.5 vs. RE_L: 5.5 ± 1.5 vs. RE_M: 6.8 ± 1.3 $mmol \cdot L^{-1}$; $p < 0.05$), DP (CON: 8,415 ± 1,223 vs. RE_L: 15,980 ± 2,007 vs. RE_M: 18,047 ± 3,693 $mm\ Hg \cdot b \cdot min^{-1}$; $p < 0.05$), and RPE (RE_L: 11 ± 2 vs. RE_M: 13 ± 2 Borg Scale; $p < 0.05$). We concluded that RE_L and RE_M were effective in reducing GLUC for individuals with T2D, with lower cardiovascular-metabolic and perceptual stress being observed for RE_L. These data suggest that acute RE sessions at light or moderate intensities are effective for controlling GLUC in individuals with T2D.

Key Words type-2 diabetes, resistance exercises, intensity, lower stress, blood glucose

Introduction

Type-2 diabetes (T2D) is a disease characterized by insulin resistance and chronic hyperglycemia, resulting in a neural and metabolic dysfunction (1) and representing a direct and independent risk factor for the development of cardiovascular complications (5). However, it has been reported that postprandial peaks of hyperglycemia are even more worrying to the onset of microvascular and macrovascular complications than elevated fasting blood glucose (GLUC) levels (5,27).

Nonpharmacological therapies such as diet and exercise have shown effective results in the prevention and treatment of T2D (10,25) and its associated complications (14,18,26). The benefits of acute aerobic exercise include insulin sensitivity (6), GLUT4 gene expression (11), and increased muscle glucose uptake throughout the subsequent 24-hour postperiod (16).

However, the acute effects of resistance exercise (RE) have been less studied than aerobic exercise (16), especially in glycemic control of individuals with T2D in postprandial moments. Despite the RE training efficiency in glucose control of individuals with T2D (2), studies comparing acute effects of different RE intensities in metabolic and hemodynamic parameters and rate of perceived exertion (RPE) are still made necessary. Thus, the purpose of this study was to compare the hemodynamic, GLUC, blood lactate (LAC),

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and RPE responses in different intensities of RE in individuals with T2D and individuals nondiabetes (ND). Our hypothesis was that both RE intensities would promote the GLUC control in individuals with T2D, however, with the lower intensity being more effective and with a less metabolic, hemodynamic and perceptual stress. This condition would be of importance to practitioners with T2D in the early stages of an RE training program. For those, a higher intensity of RE would result in hemodynamic and metabolic imbalance eliciting GLUC and blood pressure to increase.

Methods

Experimental Approach to the Problem

To test the main hypothesis, the design of this study included 3 sessions: (a) RE circuit at 23% of 1-repetition maximum (1RM) (RE_L); (b) RE circuit at 43%1RM (RE_M); and (c) control (CON) session. These sessions were performed by all volunteers in a random sequence. The GLUC, LAC, double product (DP), and RPE were measured in individuals with T2D and ND before, during, and postsessions. Comparisons among sessions and between groups were performed.

Subjects

Nineteen men volunteered to participate in the study and were divided into 2 groups: T2D ($n = 9$) and ND ($n = 10$). All the volunteers were adapted to RE. The main characteristics, clinical data, and functional capacity of the participants with T2D and ND are shown in Table 1. The individuals with T2D were diagnosed through previous medical screening by GLUC ≥ 126 mg·dl^{-1} or glycosylated hemoglobin $>7\%$ (1). The study was designed in observance of Declaration of Helsinki and was approved by the Institutional Ethics Committee. All the participants were informed of the methods before giving written consent.

Procedures and Experimental Protocol

Volunteers were instructed to attend to the laboratory in 4 distinct days. The first visit consisted of the 1RM test (13) and the other visits of 3 experimental sessions that started between 8:00 and 8:30 AM. The 1RM tests were performed in 6 different exercises (leg extension, bench press, leg press, lat pull down, leg curl, and seated row). All 3 experimental sessions were randomized and represented: (a) circuit of low-intensity RE (RE_L): consisted of 3 sets of 30 repetitions in each of the 6 exercises previously described performed at an intensity of 23% of 1RM with 2 seconds for each repetition, 15–20 seconds of rest between each exercise, and 2 minutes of rest between each set; (b) circuit of moderate intensity RE (RE_M): consisted of 3 sets of 16 repetitions in each of the 6 exercises cited above at 43% 1RM with 2 seconds for each repetition, 45–50 seconds of rest between each exercise and 2 minutes of rest between each set and; (c) CON session: same procedures used in the RE sessions but without exercise. Nevertheless, the subjects remained at a resting seated position during the period corresponding to exercise. Both the 1RM tests and RE sessions were performed in strength training equipments (Righetto, São Paulo, Brazil).

The duration of each circuit session was 25 minutes, and they had the same total workload. The exercise intensities in this study (23% 1RM and 43% 1RM) were, respectively, below and above the glucose threshold in the RE, which were previously described in *The Journal of Strength and Conditioning Research* (19). This study verified the glucose threshold at approximately 30% of 1RM during an incremental RE session in individuals with T2D, where RE performed above this threshold allowed an acute increase in the concentrations of GLUC.

Two hours before the experimental sessions (RE and CON), volunteers ingested a standard breakfast with 45 g or 180 kcal of carbohydrates, 6 g or 24 kcal of protein, and 9 g or 81 kcal of fat, totaling 285 kcal and a glycemic index of 73.5, considered as moderate (9). All individuals with T2D had treatment with diet and exercise and 5 individuals with T2D were under medication (sulfonylurea; metformin; glucovance; actos; and amaryl). Medications were maintained throughout the study.

Table 1. Mean characteristics (±*SD*) of individuals with T2D and ND.*

	T2D ($n = 09$)	ND ($n = 10$)	*p*
Age (y)	47.2 ± 12.4	50.8 ± 12.0	0.53
Weight (kg)	87.6 ± 20.0	77.4 ± 5.7	0.14
Height (cm)	175.0 ± 6.0	172.0 ± 4.0	0.23
BMI (kg·m^{-2})	28.5 ± 5.6	26.1 ± 1.2	0.21
Body fat (%)	22.4 ± 7.2	20.5 ± 3.3	0.46
$\dot{V}O_2$peak (ml·kg·min^{-1})	28.8 ± 6.6	31.6 ± 5.3	0.32
1RM total 6 exercises (kg)	574.6 ± 119.2	704.9 ± 103.8	0.02
1RM lower members (kg)	378.7 ± 90.0	480.7 ± 84.3	0.02
1RM upper members (kg)	195.9 ± 42.6	224.2 ± 40.7	0.15
FBG (mg·dl^{-1})	179.6 ± 70.1	80.3 ± 14.9	0.01
SBP baseline (mm Hg)	134.7 ± 26.8	121.5 ± 11.1	0.17
DBP baseline (mm Hg)	82.0 ± 12.1	78.2 ± 7.9	0.42
Time of diabetes (y)	4.8 ± 4.0		

*BMI = body mass index; $\dot{V}O_2$peak = peak oxygen consumption at the end of incremental test; 1RM = 1 repetition maximal; FBG = higher concentrations of fasting blood glucose measured in ambulatory conditions; SBP = systolic blood pressure; DBP = diastolic blood pressure; T2D = type 2 diabetic; ND = nondiabetic.

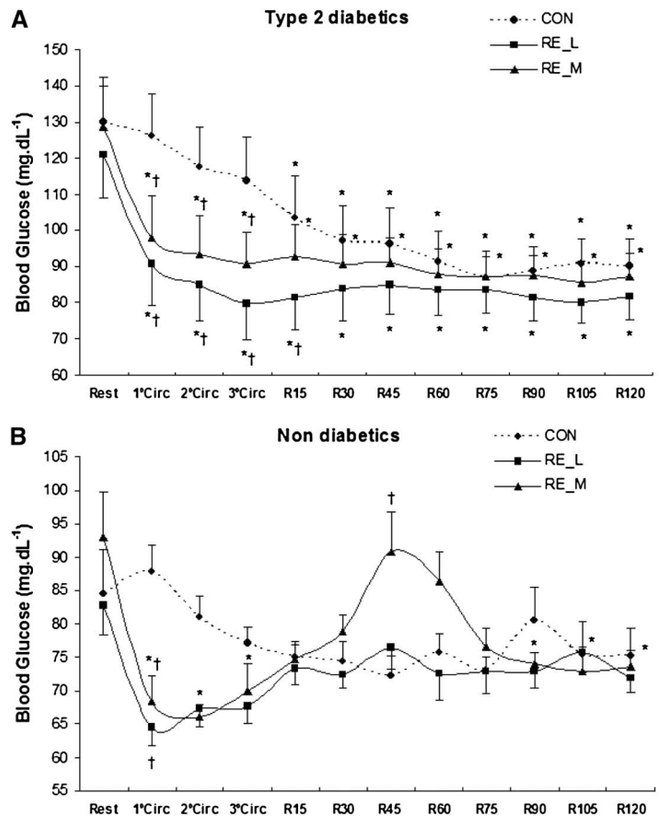

Figure 1. Mean values (±*SEM*) of blood glucose in the RE_L (23%1RM), RE_M (43%1RM), and CON sessions for individuals with type 2 diabetics (A) and nondiabetics (B). *$p < 0.05$ to rest in the same experimental session; †$p < 0.05$ to CON session in the same moment. Rest = resting preexperimental session; 1o, 2o, and 3oCirc = immediately after the end of the first, second, and third circuits of experimental sessions; R15–R120 = minutes of recovery postexperimental session; RE_L, RE_M = low resistance and moderate resistance exercise sessions; RM = repetition maximum; CON = control.

Measurements

During the experimental sessions, 25 μl of blood was collected from the ear lobe by using heparinized capillaries. Blood samples were collected in 1.5 ml microtubules (Eppendorfs) containing 50 μl of sodium fluoride at 1% and stored at −20° C for posterior analysis of LAC and GLUC concentrations (Yellow Springs Instruments 2700-Select, OH). Heart rate (HR) (Polar S-810, Kempele, Finland), systolic blood pressure (SBP) and diastolic blood pressure (DBP) were measured (Microlife BP3 AC1, Heerbrugg, Switzerland) during and postinterventions at each 15 minutes over the 120 minutes of postexercise recovery period. Double product was calculated by multiplying SBP and HR. Rate of perceived exertion (RPE) was measured using a 15-point Borg Scale (3).

With the exception of RPE, which was measured only immediately after the RE circuit sessions, the SBP, DBP, and HR were measured each 5 minutes during the 20 minutes of rest before RE and CON sessions. In addition, LAC and GLUC were only measured at the end of the

Table 2. Mean values (±*SD*) of AUC of blood glucose in individuals with T2D and ND during the 145 minutes of the experimental sessions (25 minutes of exercise or control, plus 120 minutes of postexercise recovery) in the CON, RE_L, and RE_M exercise sessions.*

		CON (no exercise)	RE_L (23%1RM)	RE_M (43%1RM)
AUC blood glucose ($mg \cdot dl^{-1}$ 145 min)	T2D	14,576 ± 3,922†‡	12,556 ± 3,269§	13,433 ± 3,054
	ND	11,498 ± 882†	10,943 ± 956§	12,156 ± 1,062

*AUC = area under the curve; T2D = type 2 diabetics; ND = nondiabetics; CON = control; RE_L, RE_M = low resistance and moderate resistance exercise sessions.
†$p < 0.05$ to RE_L and RE_M of the same group.
‡$p = 0.02$ to ND in the CON session.
§$p < 0.05$ to the RE_M of the same group.

Table 3. Mean values (±*SD*) of LAC, DP, and RPE during the 25 minutes of resistance exercise (RE_L and RE_M) and CON sessions for individuals with T2D and ND.*

		CON (no exercise)	RE_L (23%1RM)	RE_M (43%1RM)
LAC (mmol·L^{-1})	T2D	1.3 ± 0.5†	5.5 ± 1.5‡	6.8 ± 1.3
	ND	1.4 ± 0.6†	6.4 ± 1.7	7.5 ± 1.8
DP (mm Hg·b·min^{-1})	T2D	8,415 ± 1,223†	15,980 ± 2,007‡	18,047 ± 3,693
	ND	7,570 ± 1,775†	15,321 ± 3,061‡	18,433 ± 3,810
RPE (Borg scale)	T2D		11 ± 2‡	13 ± 2
	ND		12 ± 1‡	14 ± 2

*LAC = blood lactate; DP = double product; RPE = rate of perceived exertion; RE_L, RE_M = low resistance and moderate resistance exercise sessions; CON = control; T2D = type 2 diabetics; ND = nondiabetics.
†$p < 0.001$ to RE_L and RE_M of the same group.
‡$p < 0.05$ to RE_M of the same group.

20 minutes of rest. These variables were also collected between the RE circuit sets and CON sessions (3 sets × ~8.3 minutes each, totalizing 25 minutes for the entire session) and at each 15 minutes during the recovery period of 120 minutes (R15–R120) after the RE and CON sessions. Each entire experimental session lasted 145 minutes.

Statistical Analyses

Descriptive analyses (mean and *SD* or *SEM*) were presented. Student's *t*-test was applied to verify the differences in general mean characteristics between groups (T2D vs. ND). The area under the curve (AUC) was calculated for GLUC in all moments of the experimental sessions (145 minutes of total duration), being 25 minutes of RE_L, RE_M, or CON plus 120 minutes of postexercise recovery. Two-way analysis of variance (ANOVA) compared the AUC among experimental sessions at both groups (Session [RE_L vs. RE_M vs. CON] × Group [T2D vs. ND]). To compare absolute concentrations of GLUC, 2-way ANOVA (Moment [rest vs. during and postsession] × Session [RE_L vs. RE_M vs. CON]) for repeated measures was adopted. Tukey's Post hoc test was applied to detect differences in all analyses. The level of significance was set at $p < 0.05$ (Statistica version 6.0).

Results

The GLUC responses during the RE_L, RE_M and CON sessions and the 120 minutes of postexperimental recovery period (R15–R120) were compared with rest within each session. Comparisons between the same moments were also performed for the different experimental sessions, as shown in F1 Figure 1A, B (T2D and ND, respectively). It was possible to verify that in both sessions of RE, especially during the exercise, both groups showed a GLUC decrease already from the first circuit of RE (8.3 minutes; Figures 1A, B), whereas in the CON session, the decrease in GLUC for the T2D group only occurred after the recovery period (R15–R120) (Figure 1A).

Table 2 presents the comparison of AUC GLUC between T2D and ND in the RE and CON sessions during the 145 minutes of the experiment (25 minutes of RE_L, RE_M or CON plus 120 minutes of recovery). Results show that AUC GLUC in the CON session was higher in the T2D when compared with ND group ($p = 0.02$). However, no statistical difference in the AUC GLUC between T2D and ND groups was observed when they performed the RE_L and RE_M sessions ($p > 0.05$; Table 2). T2

When AUC GLUC was compared among all 3 experimental sessions in each group (Table 2), results showed statistical difference between REs vs. CON sessions ($p < 0.05$), with the RE_L session showing a lower AUC GLUC when compared with the RE_M session ($p < 0.05$).

Table 3 compared the mean values of LAC (millimoles per liter) and DP (millimeters of mercury beats per minute) during the RE or CON sessions (25 minutes) in individuals with T2D and ND. In addition, the mean values of RPE (Borg Scale) during the different RE intensities in both groups were compared (Table 3). The RE_L session promoted less increase in LAC, DP, and RPE than did the RE_M session in the T2D and ND groups (Table 3). The comparison of LAC between RE_L and RE_M in the ND group presented a trend of statistical difference ($p = 0.09$). T3

Discussion

The most important finding of this study was that RE_L and RE_M sessions were effective in reducing GLUC concentration in individuals with T2D (Figure 1 and Table 2). When the RE was performed, the results showed that individuals with T2D presented the same AUC GLUC when compared with the individuals ND (Table 2). In addition, the RE_L session showed greater benefits in GLUC control (Table 2) with lower metabolic, hemodynamic, and perceptual stress when compared with the RE_M session (Table 3).

Our results show practical applications that can be used for RE prescription in individuals with T2D. The knowledge of

RE intensities related GLUC decrease and lower metabolic and hemodynamic stress can help professional care and contribute for a safer and more effective exercise programs for this population, especially if practitioner be a beginner or have any other risk factors associated. In this sense, a moderate to low-intensity RE session would be performed in a circuit model to reduce GLUC of individuals with T2D, mainly in the first weeks of training program. The prescription would be done consisting of 3 sets of 6 exercises with 15 or 30 repetitions each at an intensity of either 45 or 25% 1RM, respectively. The recovery between exercises would be between 20 and 40 seconds in which the participant should change the exercise, and the muscle groups involved would be the large ones and exercise would be performed alternating them.

The results of this study suggest that, at least acutely, the RE_L can promote greater benefits for GLUC control in individuals with T2D than RE performed at higher intensities. This result corroborates with the study of Manders et al. (16) that shows that a single bout of aerobic exercise at a low intensity, as opposed to high intensity, effectively lowers the prevalence of hyperglycemia and, as such, improves daily glycemic control in individuals with T2D.

One clinical significance of GLUC reduction is related to glucotoxicity (20), a phenomenon that when associated with increased intracellular fat, can augment the production of reactive oxygen species (15). Daily chronic or intermittent hyperglycemia (7), oxidative stress (15), and other factors (17) can imply in structural and functional impairments to pancreatic beta cells and insulin-related tissues. In our study, GLUC analysis from the first set of RE circuit (8.3 minutes of exercise; Figure 1) and AUC from RE sessions (145 minutes; Table 2) showed significant decreases, allowing us to indicate that RE_ at a low or moderate intensity can be protective against the deleterious effects of glucotoxicity.

One of the mechanisms involved in the maintenance of near to normal GLUC levels for at least 2 hours immediately after physical exercise could be increased insulin sensitivity. Fluckey et al. (8) investigated GLUC and insulin sensitivity responses in individuals with T2D after a single RE session with intensities >50% of 10RMs. The authors identified a decrease in insulin levels until 18 hours after exercise. However, this was not accompanied by a decrease in GLUC concentration. One limitation of this study was that we did not measure insulin levels in different moments of the experiment, though, it is believed that the GLUC reduction during the 145 minutes of the experimental sessions in both RE sessions has been followed by an improvement of insulin sensitivity (8), mediated by the higher phosphorylation of GLUT4 proteins caused by muscle contraction during physical exercise in individuals with T2D and ND (11).

AU4 A more intense exercise session can inhibit the hexokinase and limit the influx of glucose phosphorylation (22). Still, glycogen phosphatase and phosphofructokinase, which control glycogen and glucose degradation via AMPK (21,23), have their activities inhibited by intramuscular pH decrease (24). Based on this, it is possible that a smaller amount of GLUC was used when exercise was performed above the glucose threshold (19), contributing to a nondecrease of GLUC in Fluckey et al.'s study (8) and to a smaller decrease of GLUC in the RE_M session of this study.

Another explanation for the differences in AUC GLUC between the RE intensities performed could be related to higher nervous sympathetic activity in the RE_M session, with subsequent strong counterregulatory hormonal responses (12) and the higher activity of glycogenolysis and gluconeogenesis (4). Therefore, a single RE session with lower physiologic stress (RE_L, Table 3) seemed to enable for a higher uptake and a lower production of GLUC during an experimental session (Table 2). It is important to state, however, that other acute physiological benefits of RE (i.e., postexercise blood pressure reduction) were revealed to be more pronounced after higher intensities (18,26).

Finally, our results support the importance of RE_L and RE_M intensities as a nonpharmacological control of blood pressure for individuals with T2D. Especially, the RE_L seemed to elicit a better GLUC control with lower physiological stress as indicated by lower LAC, DP, and RPE responses.

In summary, the RE (RE_L and RE_M) sessions performed were effective in promoting GLUC control in individuals with T2D. The RE_L session promoted a better GLUC adjustment with less metabolic and hemodynamic stress and a lower RPE when compared with RE_M in both individuals with T2D and individuals with ND. The effects of RE training at different intensities (low, moderate, and high) on GLUC responses and associated variables (cardiovascular, metabolic, and perceptual stress) still need to be better investigated in individuals with T2D.

Practical Applications

The results of this study have practical applications, especially for the early stages of a resistance training program in individuals with T2D. Individuals with T2D starting a training program may be unbalanced in the disease and present increased GLUC and blood pressure. The findings of this study revealed a single session of RE to be effective for acute control of GLUC in individuals with T2D. Thus, a moderate to low-intensity RE would be performed in a circuit model to reduce GLUC of individuals with T2D. The prescription would be done consisting of 3 sets of 6 exercises with 15 or 30 repetitions each at an intensity of either 45 or 25% 1RM, respectively. Alternatively, the RE session would also be controlled by using the RPE between 11 and 13 on the Borg Scale of 15 points. The recovery between exercises would be between 20 and 40 seconds in which the participant should change the exercise, and the muscle groups involved would be the large ones and exercise would be performed alternating them (preferably). These recommendations may have practical applications for acute GLUC control for individuals

with T2D with similar characteristics to the participants of this study.

Despite the practical suggestions given to low and moderate intensity RE for individuals with T2D, it is possible for those patients to obtain adequate GLUC and hemodynamic control during the training throughout time. Therefore, it is important for a coach to modify the requirement, being able to work with higher intensities of RE (e.g., ≥70%1RM) depending of the progression of the client. Thus, in addition to GLUC control, muscle mass and strength could also be increased in individuals with T2D as a result of training intensity progression.

Despite those exercise intensities being considered low to moderate and thus secure in terms of cardiovascular and endocrine stress, a previous medical screening, including an orthopedic, cardiovascular, and metabolic evaluation, is strongly recommended, especially to RE sessions at higher intensity.

Acknowledgments

The authors are grateful to CNPq (proc. 474328/2010-4) for the financial support. Also, their acknowledgments are extended to the Graduate Program on Physical Education and Health, Catholic University of Brasilia (UCB).

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